including 42.9% genotype 1, 14.3% genotype 2, 19.0% genotype 3 and 23.8% genotype 6. Genotype 1 was predominant in Beijing, while genotype 6 was mainly detected in Guangdong. The levels of IFN-γ production and antibody reactivity from chronic infection donors were significantly higher than those from recovery and false positive infection donors ($p < 0.001$), but no statistical difference of IFN-α among populations from three HCV infection statuses was observed.
Conclusion: IFN-γ but IFN-α may play a role paralleled with antibody in viral clearance during natural history of HCV infection.

PP-149 Seroprevalence of anti-HCV in children living in Ulaanbaatar, Mongolia

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Background: Our previous study established that HCV infection is wide-spread in Mongolia. So, many Government and non-Government measures on HCV prevention are non-specific. So, there is needed to check the results of the measures.
Objective: To assess prevalence of HCV infection in Mongolian children.
Methods: We randomly selected 199 "healthy" children (ages 1–15) in May 2009, from Ulaanbaatar, matched by age and sex and tested serum of all subjects for anti-HCV.
Results: Eighteen of 193 serum samples tested positive for anti-HCV (9.3%). Older age was more likely to show a positive test (presented in the table by age and sex).

Table 1. Anti HCV test results by age and number of subjects

Age	Subjects		Anti-HCV		%
	n	male	n	male	
1	21	12	0		0
3	24	14	0		0
5	28	14	3	2	10.7
7	26	12	0		0
9	26	13	4	3	15.4
11	23	12	3	2	13.0
13	23	10	3	1	13.0
15	22	12	5	2	22.7
Total	193	99	18	10	9.3

Discussion and Conclusion: More half the population of Mongolia lives in Ulaanbaatar. We have carried two studies on the prevalence of HCV infection in children in Ulaanbaatar (1998, 2002). These studies did not showed a significant difference in HCV seroprevalcne between rural and urban subjects. As the results, we estimate the prevalence of HCV infection in Ulaanbaatar to the figure for the whole Mongolian population. The seroprevalence, found in this study is significantly higher compared with our previous study. Therefore, all measures to help prevent HCV infection in Mongolia should be intensified.

PP-150 Evaluation of serum TIMP-1 & TGF-β1 in Egyptian patients with chronic hepatitis C, genotype 4 & chronic hepatitis B infection

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Background: HCV & HBV infections are known to be major public health problem in Egypt. As chronic liver disease progresses, an imbalance occurs between synthesis and breakdown of extra cellular matrix (ECM). Matrix metallproteinases (MMPs) are involved in degrading ECM while tissue inhibitors of metalloproteinases (TIMPs) prevent their fibrolytic action. Transforming growth factor β1 (TGF-β1) is a key mediator of liver fibrogenesis. Aim of this study is evaluating TIMPs & TGF-β1 as fibrogenic seromarkers in comparison to histopathological examination of liver.
Methods: Commercially available ELISA assays were used to study circulating levels of TIMP-1 & TGF-β1 in 50 patients with chronic hepatitis C patients and 20 patients with chronic hepatitis B compared to 10 healthy control subjects.
Results: In HCV patients, TIMP-1 was elevated with more progresive liver disease with a sensitivity of 100% the specificity of 87%. Serum level of TIMP-1 in chronic hepatitis (B) was slightly decreased from healthy control without significant correlation. Its mean and standard deviation was (157.81±44.1) and (164.18±42.22) in the HBV and control group respectively.
In comparison between HCV & HBV patients there was significant increase in serum level of TIMP-1 in HCV patients than HBV patients.
Serum levels of TGF-β1 were decreased in HCV patients significantly from healthy control group (mean 138.27±47.8) to F4 patients (mean 46.97±23.49). Serum levels of TGF-β1 in chronic hepatitis B patients showed no statistical difference from the healthy control. Its mean and standard deviation in HBV group and control group were (114.9±53.11 and 138.37±47.86) respectively.
Conclusion: TIMP-1 is a reliable predictor to detect advanced fibrosis, in HCV patients genotype 4.

PP-151 Increased PD-L1 expression and PD-L1/CD86 ratio on dendritic cells were inversely associated with impaired dendritic cells function in HCV chronic infection

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Hepatitis C virus (HCV)-specific T cell immunity was impaired in HCV chronic infection and resulted in the persistence of HCV infection. Dysfunction of dendritic cells (DCs) was believed to be involved in this T cell exhaustion, but the mechanisms were rarely understood.
In this study, we evaluated surface costimulatory marker (CD83, CD86, and CD40) and coinhibitory marker (PD-L1) expression and allostimulatory capacity of plasmacytoid DCs and myeloid DCs in twenty five HCV-infected patients compared to twenty healthy controls. All patients and control were seronegative for anti-HIV and anti-HBV. DCs were isolated from PBMC by use of BDCA-1 positive cell isolation kit for myeloid DCs and BDCA-4 positive cell isolation kit for plasmacytoid DCs according to the manufacturer's instructions.
We found both costimulatory marker and coinhibitory marker expression were increased in HCV infection compared with healthy control. The ratio of PD-L1 versus CD86 was also increased in HCV-infected patients and the ratio positive correlated with PD-L1 expression on DCs. Allostimulatory capacity of DCs was impaired in HCV infection and this allogeneic MLR was inversely correlated with PD-L1 expression and PD-L1/CD86 ratio.
These findings suggested that the effect of inhibitory marker PD-L1 overwhelmed the effect of costimulatory markers and negatively regulated DC-T activation. Our finding will be helpful to understand the mechanism of dysfunction

of DCs in HCV infection and shed light on the DC-based immunotherapeutic strategy.

PP-152 A novel model predicting mortality of the hospitalized patients with acute-on-chronic hepatitis B liver failure

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Background: Liver failure usually progresses into a poor mortality. Despite their accuracy, classic models including model for end-stage liver disease (MELD) and Child–Pugh score (CPS), are mainly being accepted to determine the prognosis of patients with end-stage liver disease, However, these models are still controversial in predicting mortality of patients with acute-on-chronic hepatitis B liver failure (ACHBLF). In this study, we aim to evaluate the possibility to better predict mortality of the hospitalized patients with ACHBLF using a novel logistic regression model (LRM).
Methods: The LRM was constructed using data from 242 consecutive patients with ACHBLF at Liver Research Center, Wenzhou, China (internal cohort). The LRM was tested on 210 patients listed for medical treatment at Tianjin Infectious Disease Hospital, China (external cohort). The receiver operating characteristic curves (ROCs) were drawn for LRM, MELD, CPS and Sun's model. Predictions of mortality obtained with four models on the same datasets were compared using areas under ROC curves (AUC).
Result: In internal cohort, there were 75 patients who died in hospital (31.0%). The mean MELD score for the patients who died was significantly greater than those who survived (26.8 vs. 22.4, $p < 0.001$). The LRM performed excellent diagnostic accuracy significantly better than MELD, CPS and Sun's model both in the internal cohort (AUC = 0.873 vs. 0.694 vs. 0.718 vs. 0.786; $p < 0.001$, $p < 0.001$, $p = 0.002$, respectively) and in the external cohort (AUC = 0.844 vs. 0.775 vs. 0.601 vs. 0.753; $p = 0.035$, $p < 0.001$, $p = 0.004$, respectively).
Conclusion: Our newly established LRM was superior to MELD, CPS and Sun's model in predicting mortality risk of hospitalized patients with ACHBLF.

PP-153 Acute fatty liver of pregnancy: report of 27 cases

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Objective: To explore the clinical profile, biochemical findings, complications and maternal outcome in patients of acute fatty liver of pregnancy (AFLP).
Methods: Patients with AFLP hospitalized in Beijing Ditan Hospital from Sep 1996 to Nov 2010 were analyzed. The clinical feature, laboratory examination, and treatment were retrospectively analyzed.
Results: There were 27 women included in this study with a mean age of 27.7±3.3 years (23 to 34 years). The mean gestational age was 35.9±2.2 weeks. There were 3 cases death (11.1%). The most common symptoms at admission were jaundice (20 cases), fatigue (16 cases), nausea and vomiting (13 cases) and pruritus (5 cases). During hospital stay, three patients presented psychiatric symptom, manifesting as visual hallucination, auditory hallucination, and delusion of persecution. Laboratory examination: WBC increased, anemia, thrombocytopenia, hypoalbuminemia, the total bile acid increased, coagulation dysfunction (PTA < 80%) and renal injury. Compared with non-ALF group (n = 14), the ALF group (n = 13) patients have higher white blood cell, TBIL, DBIL, Cr lever and lower hemoglobin and PTA ($P < 0.01$). The ALF group patients were more likely to present hypoglycemia, acute renal failure, coagulation disorders, acute respiratory distress syndrome, neonatal asphyxia, post-partum hemorrhage and fetal death ($P < 0.05$). There are 24 cases discharged home with full recovery of hepatic and renal function. Other three cases were dead. The causes of death were cerebral hemorrhage, post-partum hemorrhage, hemorrhagic shock and multiple organ failure.
Conclusions: (1) Patients with ALF are more likely to occur multiple organ dysfunction, asphyxia neonatorum and fetal death. (2) With adequate support, the most patients may have full recovery of hepatic and renal function.

PP-154 Alpha fetoprotein is a novel inhibitor of apoptosis protein mediated human hepatoma Bel 7402 cells resisting the apoptosis induced by TRAIL

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Objective: AFP has a property to maintain hepatocellular carcinoma cells (HCC) growth in vivo, and hepatitis B virus X (HBX) protein up-regulates AFP expression in HCC, but the critical function of AFP was unclear, the present investigation explore the influence of AFP on the apoptosis induced by tumor necrosis factor-related apoptosis-inducing ligand (TRAIL) of HCC.
Methods: Co-localization and interactIon of AFP and caspase-3 or caspase-8 observed by confocal microscopy or co-immunoprecipitation (Co-IP); siRNA was applied to knockdown the expression of AFP in Bel 7402 cells (AFP-producing); The activity of caspase-3 or caspase-8 was detected by protease activity colorimetric methods; Flow cytometry and MTT were used to analyze apoptosis and growth of the cells; and fluorescent microscopy was used to observe the apoptosis of Bel 7402 cells.
Results: AFP harbors a function to interact with caspase-3 in cytoplasm, but could not binding with caspase-8; The activity of caspase-3 or caspase-8 was enhanced and the expression of AFP was repressed when treated with all trans retinoic acid (40 μmol/L) plus TRAIL (2nmol/L); Treated with TRAIL (2nmol/L) alone, the activity of caspase-8 was promoted and activity of caspase-3 has not any alteration; AFP was able to inhibit the activity of caspase-3 but it has any effect on the activity of caspase-8 in vitro; Bel 7402 cells resisted the apoptosis induced by TRAIL (2nmol/L); While knockdown AFP expression by siRNA followed treatment with TRAIL (2nmol/L), caspase-3 activity was increased but caspase-8 activity had not any change, and Bel 7402 cells growth were inhibited (ratio 48.4%), and TRAIL could induce the apoptosis of the cancer cells.
Conclusions: AFP inhibits caspase-3 activity is the pivotal events that AFP mediated HCC resists apoptosis induced by TRAIL.